Environment Canada Environnement Canada

Guidelines for Surface Water Quality

Vol. 2 Organic Chemical Substances

PHTHALIC ACID ESTERS

The first draft of this document was prepared under contract by M. Lupp and L.S. McCarty of MacLaren Plansearch Inc. in 1982.

INLAND WATERS DIRECTORATE
WATER QUALITY BRANCH
OTTAWA, CANADA, 1983

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Cat. No. En 36-432/2-1983E

ISBN 0-662-12727-7

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Abstract

A literature survey was carried out on the toxic effects of phthalic acid esters on human health, aquatic life, plants and livestock. The information is summarized in this publication. From it, maximum concentrations in water of phthalic acid esters at which toxic effects will not appear are recommended.

Résumé

On a effectué une étude de la documentation portant sur les effets toxiques des phtalates ou esters de l'acide benzènedicarboxylique-1,2 sur les humains, les organismes aquatiques, les plantes et le bétail. Ces données sont résumées dans cette publication. En se basant sur ces données, on propose des valeurs des concentrations maximales des phtalates ou esters de l'acide benzènedicarboxylique-1,2 dans l'eau auxquelles on n'observe pas d'effets toxiques.

Acknowledgments

The Water Quality Objectives Division greatly appreciates the assistance of many dedicated scientists located throughout federal agencies, especially Health and Welfare Canada, Agriculture Canada, the National Research Council of Canada, Environment Canada and Fisheries and Oceans.

Special thanks are expressed to L. Lockhart, M. Gilbertson and P. Hodson (Fisheries and Oceans), and to D. MacGregor, J. Carey and W. Strachan (Environment Canada). This report was edited by M.C. Taylor, Inland Waters Directorate.

RECOMMENDED WATER QUALITY OBJECTIVES FOR PHTHALIC ACID ESTERS

Uses	Recommended objective
Raw public water supply	No recommended limit
Aquatic life and wildlife	
Aquatic life	DBP: not to exceed 4.0 μg/L DEHP: not to exceed 0.6 μg/L Other phthalates: not to exceed 0.2 μg/L
Wildlife	Insufficient information
Agricultural water supply	
Lifestock watering	Insufficient information
Irrigation	Insufficient information
Recreation and aesthetics	No recommended limit
Industrial water supplies	
Food processing	No recommended limit

Guidelines for Surface Water Quality

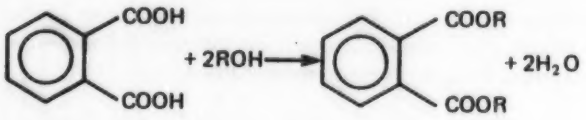
Vol. 2. Organic Chemical Substances. Phthalic Acid Esters

SOURCES, OCCURRENCE AND CHARACTERISTICS

Production and Uses

Phthalic acid esters (PAEs) or esters of 1,2-benzenedicarboxylic acid represent a large family of organic chemicals widely used as plasticizers, primarily in the production of polyvinyl chloride (PVC) resins (Leah 1977).

Phthalic acid esters are prepared by reacting phthalic acid with a specific acid to form the desired ester:



R = alkyl group

Industrially, the esters are prepared from phthalic anhydride rather than from the acid (Leah 1977).

On a worldwide scale, 1.4×10^9 to 1.8×10^9 kg (three to four billion pounds) of PAEs are produced annually (USEPA 1980). In 1977, Canadian production of PAEs approximated 22.7×10^6 to 27.2×10^6 kg (50 to 60 million pounds) (Martec Limited 1979) which translates to less than 2% of the worldwide production. In comparison, U.S. production in 1977 amounted to roughly 550×10^6 kg (1.2 billion pounds) (USITC 1978) or 30% to 40% of the world's supply.

In 1973, the last year for which detailed analyses of production and use of PAEs are available, the total supply of phthalate esters in Canada was about 35.6×10^6 kg (78.5 million pounds) (Leah 1977). Of that total, 27.9×10^6 (61.5 million pounds) was produced in Canada and 7.7×10^6 (17 million pounds) was imported (Table 1). As well, an estimated 5.2×10^6 kg (11.5 million pounds) of PAEs was contained in imported vinyl compounds, film and sheet, and an additional 6.4×10^6 kg (14 million pounds) was contained in imported fabricated vinyl products (Leah 1977).

Table 1. Canadian Supply and Demand for Phthalate Esters, 1973

	Phthalate ester (10^6 kg)	Percentage of total
Supply		
Domestic production	27.9	78.3
Imports	7.7	21.7
Total	35.6	100.0
Demand		
(i) Plasticizers for:		
Plastisols	11.1	31.2
Extruded film and sheet	10.9	30.6
Flooring	5.8	16.3
Wire and cable insulation	4.7	13.2
Other vinyl extrusions and mouldings	2.0	5.6
Adhesives	0.2	0.6
Cellulose film coatings	0.2	0.6
(ii) Other applications (non-plasticizer use)	0.3	0.8
(iii) Export	0.2	0.6
Total	35.6	99.5

Source: Leah 1977

Of the total Canadian demand (Table 1), over 98% of the PAEs was used as plasticizers to increase the workability or flexibility of plastic compounds. Of this plasticizer demand, over 60% was used for plastisols[1] and extruded film and sheet. Non-plasticizer applications, such as PCB substitutes in electrical capacitors, insect repellent formulations, defoaming agents during the manufacture of paper and paperboard, cosmetics, fragrances, munitions, lacquers, decorative inks, industrial and lubricating oils, and pesticide carriers, account for less than 1% of the total PAE demand. Although these non-plasticizer applications represent only a small portion of the total demand, the PAEs used in their manufacture are more readily released to the aquatic environment, since they are not incorporated into the matrix of a polymeric structure such as PVC (Pierce *et al.* 1980).

[1]Plastisols are paste-like suspensions of PVC polymer particles in plasticizers. They are used for "spread" coatings on textiles and paper (e.g. vinyl wall papers), dipping operations (e.g. dish draiers) and slush moulding (e.g. footwear, toys).

Table 2. Phthalic Acid Esters – Estimated Use Patterns in Canada

Compounds	Abbreviation	Use
Dioctylphthalate*	DOP	Most common PVC plasticizer
Di-(2-ethylhexyl)phthalate	DEHP	Film production mostly; also in wire, cable and adhesive
Di-*iso*-decylphthalate*	DiDP	80% in wire and cable; 20% auto vinyl upholstery
Dibutylphthalate	DBP	70% adhesives, consumer; 30% plastisols
Dicyclohexylphthalate*	DCHP	Heat sealer for cellophane
Diethylphthalate	DEP	Adhesive for cellulose and PVC
Diundecylphthalate*	DUP	100% wire and cable coatings
Butylbenzylphthalate	BBP	90% vinyl flooring (rolled) and tiles; 10% footwear and raincoats
Diheptyl-*n*-nonylundecyl-phthalate	DH*n*NDP	90% automotive exterior plastics; 10% industrial pool
Butyloctylphthalate*	BOP	Plasticizer PVC
Dimethylcyclohexyl-phthalate*		Pressure-sensitive adhesives
Di-*iso*-octylphthalate	DiOP	Plastisols
Dimethylphthalate	DMP	General plasticizer bug repellent
Ditridecylphthalate	DTDP	Plasticizer PVC

*Manufactured in Canada
Source: Martec Limited 1979

Table 3. Production of Phthalic Acid Esters in the United States in 1977

Ester	Production (10^6 kg)*	Percentage of total
Dibutyl	7.390	1.4
Diethyl	7.925	1.5
Di-*iso*-decyl	72.832	13.4
Dimethyl	4.485	0.8
Dioctyl		
Di-(2-ethylhexyl)	176.240	32.3
Other dioctylphthalates	5.291	0.9
Ditridecyl	10.559	1.9
n-Hexyl-*n*-decyl	6.886	1.3
All other phthalate esters	253.662	46.5
Total	545.405	100.0

*Converted from pounds
Source: United States International Trade Commission 1978 cited in USEPA 1980

The estimated major end-use patterns of PAEs are presented in Table 2. The most widely used phthalate plasticizers are di-(2-ethylhexyl)phthalate (DEHP) and its isomer dioctylphthalate (DOP). (The two names are often used synonymously.) Of the 14 esters listed in Table 2, DOP, diheptyl-*n*-nonylundecylphthalate, butylbenzylphthalate (BBP) and di-*iso*-decylphthalate (DiDP) account for approximately 80% of the Canadian market (Pierce *et al.* 1980). The production of PAEs in the United States, for which more detailed information is available, is shown in Table 3. Given the close relationship between Canada and the United States, both economically and physically, the information presented in Table 3 is relevant to the discussion of PAEs in Canada.

A list of abbreviations and nomenclature for some PAEs is given in the Appendix.

Chemical Characteristics

Phthalic acid esters are relatively odourless, colourless, viscous liquids with high boiling points and low volatilities (Pierce *et al.* 1980). The physical and chemical properties of some PAEs are summarized in Table 4.

The volatility of PAEs decreases with increasing length of the alcohol side chain. Among those PAEs having isomeric side chains, volatility increased with the degree of side-chain branching (Darby and Sears 1969, Graham 1975 cited in Pierce *et al.* 1980).

While PAEs are generally not very soluble in water, they are readily extracted by numerous common organic solvents. Solubility is dependent upon the length and configuration of the ester side chain, with the short-chain esters being more soluble in water (Pierce *et al.* 1980).

Phthalic Acid Esters in the Environment

With the exception of those quantities destroyed by combustion, all PAEs produced, imported and used in Canada will eventually be released into the environment as PAEs or degradation products. The release of PAEs to the environment may be broadly classified into two categories (Leah 1977):

1. *Losses of PAEs from production and processing activities* including production of PAEs, the compounding of plasticizers and PVC resin, the fabrication of flexible PVC products and the production of adhesives, plastisols and other miscellaneous products.

Table 4. Physical and Chemical Properties of Some Phthalate Esters

Esters	Molecular weight	Specific gravity (25°C)	Melting point (°C)	Boiling point (°C) (at 760 mm Hg)	Vapour pressure (mm Hg)	Water solubility (g/L at 20°C–25°C)	Solvent solubility	Log P_{ow}* (estimate)
Short-chain								
Dimethyl	194.2	1.188–1.192	0.0 (freezes)	147 (10 mm) 282	1.1×10^{-3}	5.0	Insoluble in petroleum oils; soluble in all other common organic solvents and oils	1.3
Diethyl	222.3	1.115–1.119	–40.5	158 (10 mm) 296	4.0×10^{-4}	1.0	Soluble in all common organic solvents and oils	1.8
Diallyl	246.3	1.120	–	290	–	0.1		–
Diethylene glycol	254.2	1.24	–	–	–	–	Soluble in methanol, ethanol	–
Ethyl phthalyl ethyl glycolate	269.3	1.18	–	–	–	0.18	Soluble in most common organic solvents	–
Di-*n*-butyl	278.4	1.044–1.048	–35	192 (10 mm) 340	1.4×10^{-5}	4.5 2.2	Soluble in all common organic solvents and oils	2.2
Di-*iso*-butyl	278.4	1.040 (20/4)	–50	327	–	0.1		–
Dimethoxyethyl	282	–	–	–	–	11.3 8.5		–
Long-chain								
n-Butylbenzyl	298.4	–	–	–	8.6×10^{-6} (20°C)	0.0029 ± 0.0012		4.8 ± 4.6
Dicyclohexyl	330.4	1.29	63.0	231 (10 mm)	4.5×10^{-7}	<0.1	Soluble in acetone, *n*-butanol, carbon tetrachloride, cyclohexanone, ethyl acetate, ethyl ether, toluene	3–4
n-Butyl-*n*-octyl	334.5	1.001	–50	340 (740 mm)	–	–		–
Butyl phthalyl butyl glycolate	336.4	1.093–1.103	–	219 (5 mm)	–	0.12		–
n-Butyl-*n*-decyl	362.6	–	–	–	–	–		–
Di-*n*-octyl	390.6	0.978 (20/4)	–25	220	<0.2 (150°C)	0.14 (37°C)		3–4
Di-(2-ethylhexyl)	390.6	0.990–0.985	–50	236 (10 mm) 387 (5 mm)	1.6×10^{-7}	<0.05	Miscible with most common solvents	3–4
Di-*iso*-octyl	390.6	0.981 (20/4)	–4	239 (5 mm)	1.0 (200°C)	–		3–4
n-Octyl-*n*-decyl	418.7	0.970	–28	250 (5 mm)	–	–		3–4
Di-*iso*-decyl	446.7	0.963–0.969	–48	256 (10 mm)	3.5×10^{-9}	<0.05 0.018 (37°C)	Limited solubility in glycerine, glycols and some amines; soluble in most other organic liquids	3–4
Ditridecyl	530.9	1.054	–37	235 (3.5 mm)	–	–		3–4

*Log of partition coefficient (octanol/water).

Source: Modified from Pierce *et al.* 1980. Data compiled from Darby and Sears 1969; Penn 1971; Fishbein and Albro 1972; Grasselli 1973; Graham 1975; Patterson *et al.* 1976; Clement 1977; Tomita *et al.* 1977; Monsanto 1978; Quakenbos 1953. Data from Giedhill *et al.* 1980, USEPA 1980, Lawrence 1978 and Hawley 1981 added.

2. *PAE releases at the consumer level from use and disposal activities*, which include volatilization and leaching of PAEs from PVC products during their useful lifetimes, and during and after disposal of products containing PAEs via incineration or landfill.

Very little information is available on quantities and rates of PAE losses to the Canadian environment from production or processing activities. Based on industry-supplied estimated loss rates, PAE releases to the environment in 1973 from these activities were calculated to be 0.7×10^6 to 1.7×10^6 kg (Table 5) (Leah 1977). Losses from PAE production and processing activities would be confined to the southern Ontario and mid-St. Lawrence drainage basins, where these activities are concentrated (Leah 1977; Martec Limited 1979). No actual monitoring of loss rates has been undertaken.

Phthalic acid ester losses to the environment at the consumer level are also difficult to estimate due to the absence of quantitative data on the release of PAEs from disposal via landfill, incineration or open-burning, the volatilization and leaching rates of PAEs from fabricated products during their useful lifetimes, and the time lapse between fabrication and disposal of plasticized products. Quantities of PAEs released to the Canadian environment at the consumer level in 1973 have been estimated to be 3.8×10^6 kg (Leah 1977). This represents approximately three quarters of the total national loss.

Losses from use and disposal activities, unlike those from production and processing activities, would be distributed over a greater geographic area as determined by demographic patterns. Since population densities are the greatest in the southern Ontario and mid-St. Lawrence drainage basins, the greatest consumer-related PAE losses would be expected in these areas (Leah 1977).

The release figures presented in this section represent totals only and do not indicate quantities of PAEs released directly to the aquatic environment as opposed to the atmospheric or other routes of entry. The major releases from production and processing activities are to the atmosphere, while those associated with disposal are via landfill (Leah 1977). Airborne PAEs will eventually precipitate out onto soil or directly onto water bodies. The PAEs released in landfill sites may form complexes with some components of the soil or may be carried by runoff or groundwater to adjacent water bodies. Based on very limited data, the total atmospheric deposition of airborne DBP and DEHP to the Great Lakes has been estimated to be 95×10^3 kg/year (Eisenreich *et al.* 1981). More research into the environmental movement of PAEs is required before their ultimate fate can be established.

Table 5. Releases of PAEs from Production and Processing Activities, 1973

Activity	PAEs released (10^3 kg)
PAE production	28–84
PVC compounding and processing:	
Compounds preparation	19
Flooring products processing	59–295
Sheet and film, calendered extruded	11–33
Wire and cable extrusion	4.5–9.5
Plastisols	578–1145
Other vinyl extrusion and moulding	2
Other:	
Adhesives formulating	4
Cellulose film coating	3
Total	712–1598

Source: Leah 1977

Levels and Interactions in Water and Sediment

The ease of extraction and the widespread use of PAEs, either alone or in PVC compounds, account for their ubiquity in the environment (USEPA 1980). However, considerable uncertainty exists concerning whether PAEs measured in environmental samples are biologically synthesized (naturally occurring), anthropogenic, or whether they originate during sampling or laboratory analyses (i.e. sample contamination) (Mathur 1974). Contamination of samples has been addressed in several studies (Bauman *et al.* 1967, Etemadi 1963, Thiele and Truper 1972, cited in Leah 1977; Williams 1973, Giam *et al.* 1975, Webster and Nickless 1976 cited in Pierce *et al.* 1980). Contamination can be eliminated if suitable precautions are taken and proper controls are run (Peakall 1975).

Certain plants and animal tissues may synthesize PAEs (Peakall 1975). However, whether significant amounts of PAEs are biosynthesized is difficult to establish due to the impacts of man-made releases (Mathur 1974).

The fate of PAEs in water is not well understood. High levels found in bottom sediments in relation to the overlying water column suggest that PAEs may be absorbed to particulate material in streams and ultimately deposited in bottom sediments (Johnson *et al.* 1977). Another suggested mechanism for PAE transport is the complexing or sorption of PAEs with fulvic acid, a chemical fraction found in humic substances in water and soil (Ogner and Schnitzer 1970; Whitby and Schnitzer 1978; Khan 1980). Since the fulvic acid complex is water soluble, this mechanism would be a good vehicle for the transport of the relatively insoluble PAEs within the aquatic environment.

Table 6. PAEs in Water

Type of water	Location	Compound	Concentration (μg/L)	Source*
Surface	Black Bay, Lake Superior, Ontario	DEHP	300	Mayer *et al.* 1972
	Hammond Bay, Lake Huron, Michigan	D*n*BP	0.04	Mayer *et al.* 1972
	Lake Huron, Michigan	DEHP	5	Mayer *et al.* 1972
	Missouri River, McBain, Missouri	DEHP	4.9	Mayer *et al.* 1972
	Missouri River, McBain, Missouri	D*n*BP	0.09	Mayer *et al.* 1972
	Charles River, Boston, Mass.	Phthalate	0.88–1.9	Hites 1973
	Tama River, Tokyo, Japan	DEHP	0.5–4.4	Morita *et al.* 1974
	Tama River, Tokyo, Japan	D*n*BP	0.4–5.6	Morita *et al.* 1974
	Lake Ontario (lake-wide)	Phthalate	2–50	Strachan 1976
	Lake Erie (lake-wide)	Phthalate	0.7–6	Strachan 1976
	Lake Huron (lake-wide)	Phthalate	0.8–3	Strachan 1976
	Lake Superior (lake-wide)	Phthalate	<0.1	Strachan 1976
	Lake Michigan (2 of 13 sites)	BBP	2–4	Ewing *et al.* 1977
	Lake Michigan (3 of 13 sites)	DBP	1–14	Ewing *et al.* 1977
	Lake Michigan (10 of 13 sites)	DEHP	1–137	Ewing *et al.* 1977
	Nipigon Bay, Lake Superior (near pulp and paper mill outfall)	DOP	0.1–2	Brownlee and Strachan 1977
	St. Clair River	DBP	1	Michigan Dep. of Natural Resources 1978
	St. Clair River	DEHP	1.6–4.6	Michigan Dep. of Natural Resources 1978
Finished drinking water	30 treatment plants throughout Ontario	Phthalate	Trace–9.0	Health and Welfare Canada 1980b
Groundwater	Guelph	Phthalate	4.4	Health and Welfare Canada 1980b
	Norman, Oklahoma near landfill site	DEP	4.1	Dunlap *et al.* 1976
	Norman, Oklahoma near landfill site	D*i*BP	0.1	Dunlap *et al.* 1976
	Norman, Oklahoma near landfill site	DCHP	0.2	Dunlap *et al.* 1976
	Norman, Oklahoma near landfill site	DOP	2.4	Dunlap *et al.* 1976
Pulp and paper mill effluent	Nipigon Bay, Lake Superior	DOP	15	Brownlee and Strachan 1977
Raw drinking water sources	10 U.S. cities	DPB	Trace–5	USEPA 1975
	10 U.S. cities	DEP	Trace–1.0	USEPA 1975
	39 public water system wells, New York State	DEHP	ND–170	Kim and Stone 1980
	39 public water system wells, New York State	Di-*n*-butylphthalate	ND–470	Kim and Stone 1980
	39 public water system wells, New York State	DEP	ND–4.6	Kim and Stone 1980
	39 public water system wells, New York State	BBP	ND–38	Kim and Stone 1980

*All references cited are from Pierce *et al.* 1980 with the following additions: Health and Welfare Canada 1980b; Dunlap *et al.* 1976 cited in Health and Welfare Canada 1980b; USEPA 1975 cited in Harris 1976; Kim and Stone 1980.

Table 7. PAEs in Bottom Sediments

Type of sediments	Location	Compound	Concentration (μg/kg)	Source*
Lake	Lake Ontario near mouth of Niagara River (0–3 cm depth)	Phthalate (total)	65	Mudroch 1982
	Black Bay, Lake Superior	DEHP	200	Mayer *et al.* 1972
	Lake Superior	D*n*BP	100	Mayer *et al.* 1972
	Lake Superior (28 sites)	DEHP	N.D.–1.5	Kinkead and Chatterjee 1974
	Lake St. Clair	DEHP	3.8–5.3	Michigan Dep. Natural Resources 1974
	Nipigon Bay, Lake Superior	DOP	0.7	Brownlee and Strachan 1977
	Lake Erie, Detroit River	DBP	3–6	Michigan Dep. Natural Resources 1978
	Lake Erie, Detroit River	DEHP	1–5	Michigan Dep. Natural Resources 1978
	Munising Harbour, Michigan	Phthalate	4 100	Upper Lakes Ref. Group 1977
	Lake Michigan	DEHP	N.D.–218 000	Schacht 1974
	Lake Michigan	DBP	25 000–120 000	Schacht 1974
River	Netherlands (3 rivers)	DEHP	1 000–71 000	Schwartz *et al.* 1979
	Netherlands (3 rivers)	D*n*BP	N.D.–16 000	Schwartz *et al.* 1979
	Settleable solids from 3 Michigan rivers	DEHP	≤1 000–25 000	Michigan Water Resources Commission 1973
	Upper Saginaw River, Michigan	BBP	567 (avg.)	Gledhill *et al.* 1980
Sewage sludge	54 municipal sewage treatment plants, Michigan	DEHP	17–844	Lake Michigan Toxic Substances Committee 1974

*Modified from Pierce *et al.* 1980 with the following additions: Upper Lakes Reference Group 1977; Schacht 1974 cited in Health and Welfare Canada 1980b; Michigan Water Resources Commission 1973 cited in Leah 1977; Gledhill *et al.* 1980; Lake Michigan Toxic Substances Committee 1974 cited in Johnson *et al.* 1977; Mudroch 1982, personal communication, National Water Research Institute, Burlington, Ontario.

Tables 6 and 7 summarize levels of PAEs detected in water and sediment, respectively. The tables show that PAEs are relatively insoluble and tend to partition to sediment in the aqueous environment as would be expected from their log P_{ow} values (Table 4). As can be seen in the section on Aquatic Life and Wildlife, they also tend to partition to aquatic biota.

Persistence and Abiotic Degradation

The contribution of chemical hydrolysis to the degradation of PAEs in the aquatic ecosystem is not considered significant due to their low rates of reaction (Pierce *et al.* 1980). Based on linear free energy relationships, the hydrolysis half-lives for DMP and DEHP are calculated to be four months and over 100 years, respectively, at pH 8 and 30°C (Wolfe *et al.* 1980).

Little information is available on photolysis of phthalic acid esters; like chemical hydrolysis, however, it does not appear to be a major degradation pathway. No significant decrease in butylbenzylphthalate (BBP) concentrations in water was noted either in samples exposed to sunlight or in dark controls after 28 days (Gledhill *et al.* 1980).

Very little is known about the thermal stability of PAEs (Pierce *et al.* 1980). In one study, PAEs were found to be volatilized but not degraded during oxidative thermal decomposition at 400°C (Paciorek *et al.* 1974). Low temperature incineration and open-burning of discarded plastics may be an important indirect source of PAEs to the aquatic environment. Peakall (1975) has speculated that approximately 66% of PAEs would be destroyed by low-temperature incineration and open-burning in dumps. Air samples in the vicinity of municipal incinerators in Hamilton, Ontario, contained appreciable quantities of PAEs (300 ng/m^3 of DEHP, 700 ng/m^3 of D*n*BP) (Thomas 1973).

Typical concentrations in the air in European industrial regions were in the range of 10–100 μg/m^3 (Cautreels and Van Cauwenberghe 1976, 1978 and Bove *et al.* 1978 cited in Løkke and Bro-Rasmussen 1981). It was found that DEHP and DBP were present in both the gas phase and adsorbed on particulates (Giam *et al.* 1980, and Cautreels and Van Cauwenberghe 1978 cited in Løkke and Bro-Rasmussen 1981).

As will be seen later in this report, it appears that abiotic degradation is a minor factor; degradation by biota is considered to be a substantially more important mechanism for the breakdown of PAEs in the aquatic environment.

RATIONALE

Raw Public Water Supplies

Phthalates in the Human Body

A comprehensive report on PAEs and human health appears in documents published by Health and Welfare Canada. A maximum acceptable concentration for phthalic acid esters in drinking water has not been proposed, as concentrations in Canadian drinking water are well below the levels at which PAEs have been found to cause adverse effects in humans. A brief review is included here; more details can be obtained from Health and Welfare Canada (1980a,b).

Intake of phthalates into the human body can be from a variety of sources. Given their use in numerous plastic formulations and manufacturing processes and lesser, but still significant non-plastic applications, distribution is very widespread. Total intake of any human is dependent on the extensive variety of exposure combinations.

With the ubiquity of plastics, respiratory absorption of volatilized PAEs from plastic is common. Obviously, no typical levels exist, but Peakall (1975) reported a level of about 0.2 mg/m^3 in a closed room having PVC floor tiles. Levels at PVC manufacturing sites in the USSR have been reported to be one to two orders of magnitude higher (Milkov *et al.* 1973 cited in USEPA 1980).

Dermal uptake is expected with the direct application to the skin of cosmetics containing phthalates and, especially, insect repellants which incorporate phthalates in their formula. There is known to be ester-specific variability in rates of dermal uptake (Thomas *et al.* 1978). Direct input into the human body results from the widespread use of plastics containing phthalates in medical applications. The contribution from blood stored in standard plastic blood bags and from hemodialysis is especially noteworthy (Jaegar and Rubin 1970, 1972 cited in Lawrence and Tuell 1979).

Oral intake of phthalates is effected from several sources: phthalates leached into food from plastic wraps and containers, plant and animal-derived foodstuffs containing residual PAE from general environmental exposure, and drinking water. Drinking water may have contamination at the source as well as during transport and handling prior to ingestion, e.g. PVC water pipes and plastic storage containers (Peakall 1975; Leah 1977).

Rall (1973 cited in Peakall 1975) considers inhalation to be substantially more important than oral uptake in the overall exposure to phthalates. In contrast, Leah (1977) has generated a rough estimate of daily intake and suggests that for the average Canadian, intake from food, inhalation and water is of the order of 80, 3 and 2 μg, respectively, i.e. a total of 85 μg. Health and Welfare Canada (1980b) has estimated that in a worst case the daily intake from food, air and water would be 192, 18 and 20 μg, respectively, for a total of 230 μg. For the typical 70-kg man, this represents an intake rate of 1.2 and 3.3 μg/kg of body weight per day, respectively, for the two total intakes presented above. In terms of DEHP, the total intakes noted above represent intakes of about 0.22 and 0.59 μmoles per day. Intake from biomedical sources, dermal applications, and certain manufacturing and industrial situations are not included and would substantially increase the intake for certain groups of people (USEPA 1980). The proportion of the intake that is actually absorbed into the body is unknown.

Presently available information (Peakall 1975; Thomas *et al.* 1978; USEPA 1980) indicates that phthalates are metabolized by humans and laboratory animals and are excreted in the urine, feces and bile. Rates of metabolism and excretion, metabolites, and routes of excretion vary with the ester in question, with most of the work having been done with DEHP. The liver, pancreas and gut have substantial phthalate hydrolase activity. Once in the body the ester and metabolites are widely distributed to various organs and tissues. There are some variations in concentrations in various organs. High levels in the lungs, liver and kidney are often noted immediately after application, but there does not appear to be long-term organ specific accumulation. The half-life of phthalates in the mammalian body is short. For orally administered DEHP, the half-life in rats was reported to be of the order of 0.5 to 2 days (Lake *et al.* 1977 cited in USEPA 1980; Tanaka *et al.* 1978). Ikeda *et al.* (1978, 1980) report half-lives of di-*iso*-octylphthalate and butylglycolylphthalate to be of the order of three to four days when given orally to rats, dogs and pigs.

Despite elimination, trace levels of PAEs are detectable, probably as a result of a steady state balance with general environmental uptake. Tomita *et al.* (1977 cited in USEPA 1980) reported background blood levels for DEHP and D*n*BP of about 0.02 mg/L in humans. Jacobson *et al.* (1977 cited in USEPA 1980) reported the presence of DEHP or its metabolites in the blood of

primates up to 14 months after a DEHP-spiked blood transfusion. In a study of DEHP levels in human autopsy tissue, Chen *et al.* (1979) reported that patients not exposed to known medical sources of DEHP did not have detectable levels (limit 0.3 mg/kg wet weight). Those that were exposed via transferred blood or dialysis sporadically exhibited levels above the detection limit, in the range of 0.5 to 4.6 mg/kg, up to several months subsequent to exposure. Overturf *et al.* (1979) found levels of DBP and DEHP ranging from 0.6 to 144 μg/mg in neutral lipid fractions obtained from human kidney tissue. Donors had no records of blood transfusions.

Acute Toxicity of Phthalates

Table 8 provides a summary of the acute oral toxicity of a variety of phthalates to rats and other laboratory animals. Data on dermal application and injection generate similar LD_{50} estimates (a single dose producing 50% mortality in a group of test animals within a short period of time, usually less than two weeks).

For substances of varying molecular weights, expressing doses in terms of weight obscures the fact that differing numbers of molecules of the compounds are being introduced into the test animals. For this reason, LD_{50} doses have been converted to moles per kilogram of body weight in Table 8. Linear regression analysis (Sokal and Rohlf 1969) of the data in Table 8, with molecular weight as the fixed variable and the corresponding LD_{50} dose in moles per kilogram as the random variable, generates the following:

$$LD_{50} = 0.00031 \text{ (mol wt)} - 0.042$$

A statistically significant portion ($F = 10.06$; $F_{99(1,11)} = 9.33$) of the variance can be attributed to the regression. A test of the regression coefficient indicates that the slope is significantly different from zero ($T = 3.16$; $T_{99(11)} = 3.106$). This is to say that over the molecular weight range tested, the lowest weight ester is about 6.5 times more toxic than the highest, on a molar basis. Based on the regression and converting from rat data using the USEPA (1979) method for a 70-kg man, the estimated human LD_{50} values for DMP, DEHP and DTDP are 48 g (0.25 moles), 421 g (1.08 moles) and 872 g (1.65 moles), respectively.

Differences in phthalate water solubility and octanol/water partition coefficients likely influence toxicity (Autian 1973; Lawrence 1978). Generally speaking, relative polarity, and hence water solubility, is an inverse function of the length of the carbon side chains; solubility and toxicity, however, also are related to the exact nature and structure of the side-chain groups. As can be seen from Table 4, the limited and relatively crude estimate of solubility and octanol/water partition coefficients (log P_{OW}) effectively precludes a rigorous exploration of the significance of this aspect of the chemical character of phthalate acid esters. Further research is warranted.

Table 8. Acute Oral Toxicity of Selected Phthalates to Laboratory Animals

Ester	Molecular weight	Rat LD_{50} values* (g/kg body weight)	Rat LD_{50} (moles/kg body weight)	Range of animal values† (g/kg body weight)
Dimethyl	194	8.1 (1)‡	0.0423	2.9–8.7 (4)‡
Diethyl	222	9.2 (1)	0.0414	1.0–9.2 (2)
Diallyl	246	1.7 (1)	0.0069	0.8–1.7 (2)
Dibutyl	278	12 (4)	0.0430	8.0–23 (7)
Di-*iso*-butyl	278	20 (1)	0.0719	>13–20 (2)
Dimethoxymethyl	282	>4.4 (1)	>0.0156	1.6–6.4 (3)
Butylbenzyl	298	4 (1)	0.0134	4 (1)
Dihexyl	334	33 (3)	0.0988	29–39 (3)
Diethylhexyl	391	30 (3)	0.0767	>13–>128 (6)
Dioctyl	391	37 (2)	0.0946	30–44.2 (2)
Di-*iso*-nonyl	419	>10 (1)	>0.0239	>10 (1)
Di-*iso*-decyl	447	>62 (1)	>0.139	>62 (1)
Ditridecyl	530	>68 (1)	>0.128	>68 (1)

* Some values have been converted from milligrams per kilogram via mean specific gravity estimates
† Includes rat, mouse, guinea pig and rabbit data
‡ Number of reports used to generate values is given in parentheses
Source: Modified and edited from Peakall 1975

Sub-acute and Chronic Toxicity of Phthalates

Table 9 outlines sub-acute and chronic oral toxicity information for some phthalates. Information on non-oral application of these and various other phthalates is available but is not directly applicable as the route of introduction (dermal application and intravenous and intraperitoneal injection), and the use of carrier compounds cannot be easily related to water as the source of the toxicant. There are also differences in frequency and duration of exposure.

A general summary of all sub-acute and chronic symptoms observed for a number of esters under a variety of conditions follows. A primary target in laboratory animals appears to be the liver; its enlargement is one of the most consistent symptoms, with changes in the activity of certain enzymes, changes in the microstructure of hepatocytes and inhibition of lipid metabolism also being observed. Enlargement of the kidney is common, and changes in appearance of morphology as well as hematuria have been reported. Testicular atrophy with accompanying tissue damage has been noted as a common symptom. It is felt that especially for DEHP, DBP, DHP and dipentyl phthalate administration, testicular damage is a result of decreased body zinc levels caused by increased urinary zinc excretion. One of the most common symptoms of phthalate exposure is a reduction in growth rate and occasionally even loss of weight. Finally, swelling of the heart and brain and increased gamma globulin levels and decreased food ingestion have been reported. Although only studied a few times, it appears that symptoms disappear after cessation of administration, suggesting that these chronic effects are reversible (Morton 1980; Foster *et al*. 1980; Kawano 1980; Mushtag *et al*. 1980; Oishi and Hiraga 1980; USEPA 1980; Mangham *et al*. 1981).

From Table 9, it can be seen that there is a relatively wide variation in sub-acute and chronic toxicity levels between the results for various esters. Overall, the range in which effects have been found is from 0.26 to 11.2 millimoles per kilogram body weight per day. As a result of a more comprehensive search, Brown *et al*. (1978) reported a no-effect level for DEP about one order of magnitude lower than that of much earlier studies (Draize *et al*. 1948, and FRL Inc. 1955; both cited in USEPA 1980). Increased effort allows discrimination of very subtle effects in laboratory animals, thereby lowering the no-effect levels; the significance to the real world viability of the animals, however, becomes more and more difficult to establish. Also inter-report comparisons become virtually useless.

When all of the data of the various esters are viewed in the light of the above discussion, it is difficult to suggest that there are any ester-specific differences in toxicity that can be detected at this time. This is unlike the case for the acute toxicity information. For this reason, it will be judged that the lowest toxicity estimate will apply to all esters. For DEHP the lowest no-effect level estimate is 0.13 millimoles per kilogram body weight per day. It is felt that a value of 0.1 millimoles per kilogram per day represents a conservative estimate of a no chronic effect level for all esters.

There is some conflicting evidence of carcinogenicity, teratogenicity and mutagenicity, and further research is needed to determine its relevance to humans (Peakall 1975; Thomas *et al*. 1979; USEPA 1980; Health and Welfare Canada 1980a,b; Lewandowski *et al*. 1980; Yagi *et al*. 1980; Ruddick *et al*. 1981).

Mode of Action and Interaction with Other Compounds

Toxicological modalities of phthalic acid ester action are obscure. In the case of DEHP, it is felt that the toxic effect is largely the result of the formation of the monoester and/or other metabolic breakdown products rather than the DEHP itself (Lake *et al*. 1977). However, Ruddick *et al*. (1981) have estimated the no-effect level of mono-(2-ethylhexyl)phthalate to be about 0.05 g/kg per day (0.18 millimoles per kilogram per day) for rats. Also for rats, Chu *et al*. (1981) reported a detectable effect at 0.031 g of the monoester per kilogram of body weight per day (0.11 millimoles per kilogram per day), while no effect was found at 0.0063 g/kg body weight per day (0.022 millimoles per kilogram per day). These values are close to the range of those reported in Table 9 for the diester. The differences in toxicity observed by a number of authors may be attributed, at least in part, to the fact that the doses are not expressed in moles per kilogram body weight. The molecular weight of mono-(2-ethylhexyl)phthalate is about 281, whereas that for the diester is about 391. Therefore, in an equal dose by weight, there is almost 40% more of the monoester present on a molar basis.

There appears to be a close relationship between the rate of metabolism and acute toxicity (Rowland *et al*. 1977). As well, it appears that monoesters are absorbed more quickly by rat gut than the corresponding diester. Uptake of the mono and diesters is apparently achieved by different pathways (White *et al*. 1980).

The interaction of phthalates with other compounds is not well documented.

Recommended Limit for Drinking Water

Health and Welfare Canada (1980b) has not specified a maximum limit of PAEs in drinking water. They consider

Table 9. Sub-acute and Chronic Oral Toxicity of Selected Phthalates to Laboratory Animals

Ester	Exposure level (g/kg/day)	Exposure level (millimoles/kg/day)	Symptoms	Observed (A) or estimated (B) no effect level (g/kg/day)	Observed (A) or estimated (B) no effect level (millimoles/kg/day)	Duration of test	Animal tested and source
Dimethyl	2.0	10.3	Minor growth inhibition	(A) 1.0	5.2	2 yr*	Rat (Draize *et al.* 1948)
Diethyl	2.5	11.2	Decreased growth rate	(A) 1.3	5.8	2 yr*	Rat (FRL Inc. 1955)
	–	–	–	(A) 0.63	2.8	1 yr	Dog (FRL Inc. 1955)
	0.75	3.4	Decreased growth and food intake, increased liver, stomach, and small intestine weight	(A) 0.15 (for males) (B)≤0.15 (for females)	0.68 ≤0.68	16 weeks	Rat (Brown *et al.* 1978)
Ethyl phthalyl ethyl glycolate	2.5	9.3	Decreased growth rate and kidney damage	(A) 0.25	0.93	2 yr*	Rat (Hodge *et al.* 1953)
Dibutyl	–	–	–	(A) 0.13	0.45	1 yr*	Rat (Smith 1953)
	–	–	–	(A) 0.12	0.43	1 yr*	Rat (Piekacz 1971)
	2	7.2	Decreased body and testes weight, kidney damage and zinc deficiency created	–	–	2 weeks	Rat (Cater *et al.* 1977)
Dioctyl	0.35	0.90	Transiently lower body weight, increased liver weight	–	–	1 yr	Rat (Piekacz 1971)
Butyl phthalyl butyl glycolate	–	–	–	(A) 1.0	3.0	2 yr*	Rat (Solver *et al.* 1950)
Diethylhexyl	0.4	1.0	Decreased growth rate	(B) 0.2	0.51	90 days	Rat (Boyd *et al.* 1945)
	0.2	0.51	Increased liver and kidney weight	(A) 0.065	0.17	2 yr*	Rat (Carpenter *et al.* 1953)
	–	–	–	(B) 0.06	0.2	1 yr	Guinea pig (Carpenter *et al.* 1953)
	–	–	–	(B) 0.06	0.2	1 yr	Dog (Carpenter *et al.* 1953)
	0.18	0.45	Decreased body weight	–	–	1 yr	Rat (Nikonorow *et al.* 1973)
	0.4	1.0	Transiently lower liver and kidney weight, lower body weight, decreased food consumption	(A) 0.08	0.21	2 yr	Rat (Harris *et al.* 1956)
	0.25	0.64	Increased liver weight	(A) 0.05	0.13	7 weeks*	Rat (Daniel and Bratt 1974)
	2.0	5.1	Increased liver weight, changes in hepatocyte microstructure and liver enzyme activities	–	–	3 weeks	Rat (Lake *et al.* 1977)
	0.1	0.26	Atrophy of testicles	–	–	90 days*	Rat (Cater *et al.* 1977)
	–	–	–	(B) 0.070	0.18	For gestation period	Mouse (Shiota *et al.* 1980)

*Converted from a percentage to be checked in diet assuming a mean rat weight of 500 g and a daily intake of 5% of body weight

Source: USEPA 1980 plus Harris *et al.* cited in USNAS 1977, Piekacz 1971 cited in Peakall 1975; Brown *et al.* 1978; Shiota *et al.* 1980

waterborne phthalates not to be a problem, since the maximum level reported for finished drinking water, 9 μg/L, is well below any level at which toxic human effects are noted.

Aquatic Life and Wildlife

Levels in Aquatic Biota

Table 10 summarizes environmental levels of PAEs in aquatic biota as reported by various researchers. Generally, PAE levels reported in Table 10 are greater than those encountered in water (Table 6). The greater solubility of PAEs in blood and other lipoprotein-containing materials compared with water (Thomas *et al.* 1978) accounts for the tendency for PAEs to partition to biota in the aquatic environment. This tendency is also indicated by their log P_{ow} values (Table 4).

Accumulation and Elimination of PAEs in Aquatic Biota

Bioaccumulation of PAEs in aquatic biota occurs whenever the uptake rate exceeds the rate of degradation or elimination. Uptake of PAEs may occur directly from the water through the gills or epithelial tissue of the aquatic organism and is referred to as bioconcentration. Bioaccumulation is a broader term which refers to any process by which a chemical is taken up by tissues, whether from water or diet (Macek *et al.* 1979).

The ability of aquatic organisms to bioaccumulate PAE residues in their tissues to levels above the concentration of PAEs in their aqueous environment has been demonstrated by numerous investigators.

Table 11 summarizes bioconcentration factors (BCFs) determined in various laboratory studies for some

Table 10. PAEs in Biota Collected in Field Sampling Programs

Location	Species	Compound	Concentration (μg/kg)	Source*
Black Bay, Lake Superior	Walleye	DEHP	800	Mayer *et al.* 1972
Mississippi and Arkansas	Channel catfish	DEHP	1000–7500 (3200 mean of 40 fish)	Mayer *et al.* 1972
Fairport National Fish Hatchery, Iowa	Channel catfish	DEHP	400	Mayer *et al.* 1972
Fairport National Fish Hatchery, Iowa	Channel catfish	D*n*BP	200	Mayer *et al.* 1972
Fairport National Fish Hatchery, Iowa	Dragonfly naiads	DEHP	200	Mayer *et al.* 1972
Fairport National Fish Hatchery, Iowa	Dragonfly naiads	D*n*BP	200	Mayer *et al.* 1972
Fairport National Fish Hatchery, Iowa	Tadpoles	DEHP	300	Mayer *et al.* 1972
Fairport National Fish Hatchery, Iowa	Tadpoles	D*n*BP	500	Mayer *et al.* 1972
Fairport National Fish Hatchery, Iowa	Fish available to Canadian consumers	DEHP	0–160	Williams 1973
Straits of Mackinac and Goderich, Ontario	Burbot	Phthalate	10–100	Glass *et al.* 1977
Spirit Lake, Iowa	Yellow perch	D*n*BP, DEHP	N.D.	Stalling *et al.* 1973
Clover Leaf Lake, California	Brook trout	D*n*BP, DEHP	N.D.	Stalling *et al.* 1973
Lake Superior	Lake trout	DEP	250	Swain 1978
Lake Superior	Lake trout	DBP	1700	Swain 1978
Lake Superior	Lake trout	DEHP	150	Swain 1978
Lake Superior	Lake trout	DEP	1320	Swain 1978
Lake Superior	Lake trout	DBP	40	Swain 1978
Lake Superior	Lake trout	DEHP	440	Swain 1978

*All references cited are from Pierce *et al.* 1980 with the following additions: Stalling *et al.* 1973; Swain 1978

equatic species. These factors are defined as the mean equilibrium body burden of the phthalate divided by the mean exposure concentration to which the species was subjected.

Bioaccumulation data based on static model ecosystems with D*n*OP (Sanborn *et al.* 1975) and with DEHP (Metcalf *et al.* 1973) are given in Table 12. The concentrations of PAEs in the water rapidly decreased with time and do not permit comparisons with the values in Table 11.

Except where noted, all BCFs listed in Tables 11 and 12 were based on total ^{14}C radioactivity accumulated and do not represent the actual concentration of intact PAEs in the organism. Bioconcentration factors of 155 to 886 were reported for fathead minnows exposed to DEHP based on ^{14}C analysis (Mehrle and Mayer, 1976; Mayer, 1976), whereas actual BCFs based on direct analysis of DEHP in tissue ranged from 33% to 70% less than that from the ^{14}C measurements. The misleading estimation of concentration factors from total radioactivity is also demonstrated by the results of Sanborn *et al.* (1975). Percentages of directly analyzed DOP compared with total

Table 11. Bioconcentration Factors for Phthalate Esters in Biota (based on laboratory studies)

Species	Chemical	Bioconcentration factor*	Duration (days)	Source
Cladocera, *Daphnia magna*	D*n*BP	400	14	Mayer and Sanders 1973
Scud, *Gammarus pseudolimnaeus*	D*n*BP	1400	14	Mayer and Sanders 1973
Scud, *G. pseudolimnaeus*	DEHP	54–2680†	14–21	Sanders *et al.* 1973
Sowbug, *Asellus brevicaudus*	DEHP	14–50†	21	Sanders *et al.* 1973
Rainbow trout, *Salmo gairdneri*	DEHP	42–113	36	Mehrle and Mayer 1976
Fathead minnow, *Pimephales promelas*	DEHP	155–886	56	Mayer 1976
Fathead minnow, *P. promelas*	DEHP	91–569‡	56	Mayer 1976
Bluegill, *Lepomis macrochirus*	BBP	663	21	USEPA 1978
Bluegill, *L. macrochirus*	DEP	117	21	USEPA 1978
Bluegill, *L. macrochirus*	DMP	57	21	USEPA 1978

*Based on total ^{14}C radioactivity accumulated
†Conversion from dry to wet weight
‡Based on measured concentrations of di-(2-ethylhexyl)phthalate (after USEPA 1980).

Table 12. Bioconcentration Factors for Static Model Ecosystems

Species	Chemical	Bioconcentration factor	Duration (days)	Source
Algae, *Oedogonium cardiacum*	D*n*OP	28 500*	33	Sanborn *et al.* 1975
Algae, *O. cardiacum*	DEHP	53 890†	33	Metcalf *et al.* 1973
Cladocera, *Daphnia magna*	D*n*OP	2 600*	33	Sanborn *et al.* 1975
Mosquito (larva), *Culex pipiens quinquefasciatus*	D*n*OP	9 400*	33	Sanborn *et al.* 1975
Mosquito (larva), *C. pipiens quinquefasciatus*	DEHP	107 670†	33	Metcalf *et al.* 1973
Snail, *Physa* sp.	D*n*OP	13 600*	33	Sanborn *et al.* 1975
Snail, *Physa* sp.	DEHP	21 480†	33	Metcalf *et al.* 1973
Mosquito fish, *Gambusia affinis*	D*n*OP	9 400*	33	Sanborn *et al.* 1975
Mosquito fish, *G. affinis*	DEHP	130†	33	Metcalf *et al.* 1973

*Based on actual D*n*OP measurement
†Based on ^{14}C analysis

radioactivity in the organism were 3.32% for algae, 1.8% for *Daphnia*, 4.43% for fish, 6.8% for mosquito, and 19% for snail. These numbers indicate that the BCFs reported in Table 12 for Sanborn *et al.* (1975) would be low by 22.5 times for the fish and 55.5 times for the *Daphnia* had ^{14}C analysis been used.

In a chronic exposure study utilizing a dynamic system, Mayer (1976) investigated the effect of seven different DEHP exposure concentrations on the equilibrium concentration of accumulated DEHP in fathead minnows (*Pimephales promelas*). Observing 56-day equilibrium values, the final tissue equilibrium concentration was dependent on the concentration in the exposure water up to about 5 µg/L. The relationship was linear, with equilibrium body concentrations about 500 times the concentration in the water. Above 5 µg/L and certainly above 10 µg/L in water, the accumulated body concentration was independent of the DEHP exposure concentration, with a final equilibrium body concentration of about 3 µg/L. This suggests that either (i) the uptake rate declines with increased DEHP concentration in water, (ii) the degradation efficiency increased with increased DEHP concentration in water, or (iii) less of the highly soluble DEHP is available in solution than monitoring suggests (Pierce *et al.* 1980).

Unfortunately, accumulation rates prior to equilibrium are difficult to analyze because of the lack of data on uptake efficiency and rate of degradation. It is thought that most of the material taken up is rapidly degraded and lost. Only a small fraction actually accumulates and is itself slowly eliminated. Hence, continuous exposure is necessary for the bioaccumulation of PAEs in fish (Pierce *et al.* 1980).

Bioaccumulation has been correlated with the logarithm of the octanol/water partition coefficient (log P_{ow}), which is an indicator of the lipophilic nature of a chemical (Veith *et al.* 1980). Table 4 shows estimated log P_{ow} to range from 1.5 to 2.2 for short-chain PAEs and from 3 to 4 for long-chain PAEs. Veith *et al.* demonstrated that the log BCF for fathead minnows is linearly correlated to log P_{ow} for various organic chemicals. The coefficient was found to be correlated with the BCF derived in the bioaccumulation work of Mayer (1976) for DEHP. The correlation equation developed by Veith *et al.* suggests that the long-chain PAEs bioaccumulate 100- to 1000-fold above their concentration in water, while the short-chain PAEs have little or no bioaccumulative potential (Pierce *et al.* 1980). Data given in Table 11 for BCFs for bluegill sunfish for BBP, DEP and DMP (USEPA 1978) support this suggestion.

As indicated in Tables 11 and 12, invertebrates show a higher tendency to accumulate PAEs than do fish. This suggests that degradation is either nonexistent or very slow in invertebrates and that elimination by excretion is also slow. As separate tissue analysis was not done in these studies, it is not known whether this accumulation represents exoskeletal adsorption rather than tissue uptake. The former appears to be the case for trout eggs where 96% of the DEHP associated with the eggs was found in the chorion and the rest in the yolk (Mehrle and Mayer 1976). Furthermore, during bioaccumulation studies on marine biota, Wofford *et al.* (1981) found that empty oyster shells added to tanks containing DEHP solutions caused significant decreases in water concentration relative to controls.

Biomagnification

Biomagnification refers to the process whereby bioaccumulated chemical residues increase as the materials pass up the food web through two or more trophic levels. As there is little degradation in invertebrates, they are a rich dietary source of PAEs (Pierce *et al.* 1980). Model ecosystem studies by Metcalf *et al.* (1973) and Sanborn *et al.* (1975) show that PAEs do traverse the food web. However, PAEs are not likely to be magnified across the food chain, as metabolic degradation is fairly rapid in fish and mammals. Thus, the largest concentrations would be expected at lower trophic levels, rather than at the top of the food web which occurs with DDT (Peakall 1975). The fact that a maximum equilibrium concentration, independent of input, may exist for each organism (as in the case of the fathead minnow [Mayer 1976]) further precludes extensive biomagnification (Pierce *et al.* 1980).

The relative importance of dietary and aqueous exposure of bluegill sunfish to DEHP was quantitatively evaluated by Macek *et al.* (1979). The results of their investigation indicate that incremental steady state body burden due to dietary exposure is statistically indistinguishable from that due to aqueous exposure. Hence, it was concluded that biomagnification of DEHP in the food chain is quantitatively insignificant when compared with bioaccumulation.

Biodegradation

Among the aquatic organisms that have been shown to metabolize PAEs are the microorganisms present in water, hydrosoil and sewage sludge, several invertebrates, and several species of fish.

The various studies with aquatic microorganisms have shown that a variety of PAEs are degraded in water, hydrosoil and sewage sludge. Lewis and Holm (1981) studied rates of transformation of low concentrations of DEP by batch cultures of aufwuchs microorganisms, aquatic microbial growth attached to submerged surfaces or suspended in streamers or mats. Aufwuchs fungi, protozoa, and algae did not transform the DEP, but aufwuchs bacteria rapidly transformed the DEP.

Degradation in sludge has been partially investigated. Graham (1975) reported that 91% of DEHP and 99% of BBP introduced into a laboratory-scale activated sludge process were degraded within 48 h. Similarly, Saeger and Tucker (1973) demonstrated that 74% and 96% of the DEHP and BBP were aerobically degraded in a continuous sludge system. However, high concentrations of PAEs in municipal sewage sludge, as shown previously in Table 7, imply either that the activated sludge system is not efficient in degrading PAEs or that the raw sewage contains very large amounts of PAEs. Saeger and Tucker (1976) further report that half-lives for BBP and DEHP in river water were in the order of two days and four weeks, respectively.

Although there is laboratory evidence for some biodegradation of PAEs in hydrosoil, little is known of the dynamics of PAEs in natural sediments.

Johnson and Lulves (1975) found a significant difference between conditions and rates of degradation of DBP and DEHP in laboratory-incubated hydrosoil. Under aerobiosis, 53% of the ^{14}C-labelled DBP was degraded within 24 h and 98%, within five days. In contrast, DEHP required 14 days for 50% degradation. Under anaerobiosis, degradation of both esters was retarded. When the hydrosoil was overlaid with nitrogen, DBP was degraded only one sixth as fast, whereas no anaerobic degradation of DEHP was detected after 30 days. This would suggest that some PAEs may persist in anaerobic sediments and, therefore, important bottom-dwelling macro- and microfauna could be exposed to significant PAE residues (Ontario Ministry of the Environment 1979).

The metabolism of PAEs in fish has been studied by Mayer (1976), Melancon and Lech (1976), Sanborn *et al.* (1975) and Stalling *et al.* (1973). Reviews on this subject are available in Melancon (1979) and Pierce *et al.* (1980). Generally, results of DEHP metabolism studies *in vivo* and *in vitro* suggest that the major route of metabolism for DEHP in fish consists of hydrolysis to the monoester. The monoester may then be conjugated with glucuronic acid, or further hydrolyzed to phthalic acid, or the remaining side chain may be oxidized (Melancon 1979). Also, short-chain PAEs such as DBP are generally more rapidly metabolized than long-chain PAEs such as DEHP, both in aquatic microorganisms and in fish (Melancon 1979).

The rates of biodegradation, unlike those of abiotic degradation, appear to be very rapid in most systems studied. In 0.3% and 0.5% culture solutions, DEHP was degraded in 52 and 72 h, respectively, by the bacterium *Pseudomonas acidovorans* (Kurane *et al.* 1977a,b cited in Pierce *et al.* 1980).

In a 24-h uptake study using DEHP, Stalling *et al.* (1973) showed that after a 24-h exposure in a static test, more than 85% of the DEHP was degraded to intermediate moieties in channel catfish (*Ictalurus punctatus*). In a similar static test, Melancon and Lech (1976) found that for rainbow trout (*Salmo gairdneri*) after a 24-h exposure to 0.5 mg/L ^{14}C-labelled DEHP, 50% of the radioactivity present in the fish was localized in the bile in a number of DEHP metabolites and only 1% of the bile radioactivity was from unchanged DEHP.

Depuration half-lives ($t_{1/2}$) of less than three days were estimated for DEHP in bluegill sunfish (*Lepomis macrochirus*) (Macek *et al.* 1979). Lower $t_{1/2}$ for bluegills of between one and two days were calculated for DMP, DEP and BBP (Barrows *et al.* 1980). For fathead minnows, $t_{1/2}$ of five days for DOP (Sanborn *et al.* 1975) and seven days for DEHP (Mayer 1976) were determined. Johnson (1975 cited in Johnson *et al.* 1977) found that daphnids (*Daphnia magna*) rapidly eliminated both DEHP and DBP after exposure to concentrations ranging from 0.1 to 1.0 µg/L. The daphnids eliminated more than 50% of the original phthalate residues in 1 h and more than 90% within 24 h.

Toxicity to Aquatic Organisms

The lack of initial or sustained solubility of PAEs in water poses a problem in the toxicological analysis of these compounds. To achieve high concentrations, emulsions must be used, or solvent aids are required.

Few investigators give any consideration to this problem. As there are few methodological details provided, it is difficult to determine the actual concentrations used. Therefore in the following text, most of the concentrations used by the authors should be considered nominal (Pierce *et al.* 1980).

In addition, modifications to the biological activity of PAEs caused by changes in environmental conditions (e.g. pH, temperature) and the physical and chemical state of PAEs have not been extensively studied. Birge *et al.* (1978) found that the toxicity of DOP to goldfish, rainbow trout

and largemouth bass was not substantially different at water hardnesses of 50 and 200 mg/L as $CaCO_3$. However, Gledhill *et al.* (1980) found that 96-h LC_{50} of BBP for fathead minnows to be 2100 and 5300 µg/L for water hardnesses of 40 and 160 mg/L as $CaCO_3$, respectively.

Mayer *et al.* (1972) noted that the solubilization of DEHP by fulvic acid, which occurs widely in soils and water (Khan and Schnitzer 1972), could possibly result in a greater availability of this compound to aquatic organisms. However, no investigations have been undertaken to examine the effect of this or other chemical-modifying factors on toxicity (Pierce *et al.* 1980).

Acute Toxicity

In recent years, many researchers have investigated and reported on the acute toxicity aspects of PAEs to aquatic biota. Table 13 summarizes 96-h LC_{50} estimates reported for five phthalate esters. These studies were conducted with a relatively small diverse group of freshwater fish and invertebrate species. The greatest amount of data concerning the widest range of species is available for D*n*BP and BBP.

The acute values, with one exception, all exceed 1000 µg/L. In general, the lowest lethal concentrations were reported for D*n*BP, with 96-h LC_{50} values ranging from 730 to > 10 000. Bluegills and scud were, respectively, the most sensitive fish and invertebrate species tested with this ester.

In contrast, 96-h LC_{50} values for DEHP are at least an order of magnitude higher than those for D*n*BP. This is supported by the 96-h no-effect levels determined by Hrudey *et al.* (1976) for rainbow trout. These investigators found threshold concentrations of 500 µg/L and 200 000 µg/L for D*n*BP and DEHP, respectively (acetone-solvent). The 96-h LC_{50} values of DEHP shown in Table 13 are all greater than 10 000 µg/L for both fish and invertebrates.

Table 13. Acute Toxicity of Phthalates to Macroinvertebrates and Fish

Species	Chemical	96-h LC_{50}/EC_{50} (µg/L)	Source
Cladocera, *Daphnia magna*	BBP	92 300	USEPA 1978
Cladocera, *D. magna*	BBP	3 700	Gledhill *et al.* 1980
Cladocera, *D. magna*	DEP	52 100	USEPA 1978
Cladocera, *D. magna*	DMP	33 000	USEPA 1978
Cladocera, *D. magna*	DEHP	11 100	USEPA 1978
Scud, *Gammarus pseudolimnaeus*	D*n*BP	2 100	Mayer and Sanders 1973
Scud, *G. pseudolimnaeus*	DEHP	>32 000	Sanders *et al.* 1973
Midge, *Chironomus plumosus*	D*n*BP	4 000	Streufert 1977
Rainbow trout, *Salmo gairdneri*	BBP	3 300	Gledhill *et al.* 1980
Rainbow trout, *S. gairdneri*	D*n*BP	1 200	Hrudey *et al.* 1976
Rainbow trout, *S. gairdneri*	D*n*BP	6 470	Mayer and Sanders 1973
Rainbow trout, *S. gairdneri*	DEHP	540 000	Hrudey *et al.* 1976
Fathead minnow, *Pimephales promelas*	BBP	5 300	Gledhill *et al.* 1980
Fathead minnow, *P. promelas*	BBP	2 100	Gledhill *et al.* 1980
Bluegill, *Lepomis macrochirus*	D*n*BP	1 300	Mayer and Sanders 1973
Bluegill, *L. macrochirus*	BBP	43 300	USEPA 1978
Bluegill, *L. macrochirus*	BBP	1 700	Gledhill *et al.* 1980
Bluegill, *L. macrochirus*	DEHP	>77 000	USEPA 1978
Bluegill, *L. macrochirus*	DEP	98 200	USEPA 1978
Bluegill, *L. macrochirus*	DMP	49 500	USEPA 1978
Bluegill, *L. macrochirus*	D*n*BP	730	Mayer and Sanders 1973
Bluegill, *L. macrochirus*	D*n*BP	1 200	USEPA 1978
Channel catfish, *Ictalurus punctatus*	D*n*BP	2 910	Mayer and Sanders 1973
Crayfish, *Orconectes nais*	D*n*BP	>10 000	Mayer and Sanders 1973

Source: Adapted from USEPA 1980

Zitko (1972) observed no mortality with juvenile Atlantic salmon exposed to 10 000 µg/L of DMP and DEHP over 96 h. At this same concentration, DBP caused 100% mortality in 3 h (LT_{50}, lethal time for 50% mortality at a given concentration was 1.25 h) but no mortality occurred at 1000 µg/L in 96 h.

Chronic Toxicity

There are very few data available on the chronic toxicity of PAEs to aquatic organisms. Results of two-generation chronic studies indicated BBP was not highly toxic to *Daphnia magna* (Gledhill *et al.* 1980). Survival in the second generation and reproduction in both generations were impaired at 760 µg/L, the highest concentration tested. At 260 µg/L no effects on survival or reproduction were noted. In the controls, survival of the test organisms averaged 90% in both generations.

LeBlanc *et al.* (in preparation, cited in Gledhill *et al.* 1980) reported the results of a 30-day post-hatch fathead minnow embryo-larval study with BBP. A statistically significant ($p > 0.05$) reduction in growth was observed at 360 µg/L. Hatchability and survival remained similar to those of the controls.

Mehrle and Mayer (1976) found that the hatchability of rainbow trout eggs was not affected by continuous exposure to DEHP at concentrations of 5 to 54 µg/L. However, DEHP exposure did cause an increase in mortality of post-hatch sac fry and a decrease in total protein. At 54 µg/L, 23% of the fry died after a 24-day exposure. This concentration is 1000-fold lower than what might be expected from acute tests on larger trout. Mehrle and Mayer (1976) determined the no-effect concentration of DEHP on mortality and protein concentration to be between 5 and 14 µg/L.

Mayer and Sanders (1973) conducted a chronic test with DEHP and *Daphnia magna*. Concentrations of 3, 10 and 30 µg DEHP/L impaired reproduction by approximately 60%, 70% and 80%, respectively, after a 21-day exposure. As the concentration of 3 µg/L was the lowest concentration tested, adverse effects on reproduction may occur at lower concentrations. Since little experimental detail of the test methodology is included, however, not too much emphasis should be placed on the data (Pierce *et al.* 1980).

McKim (personal communication, Johnson *et al.* 1977) reported that the growth of brook trout (*Salvelinus fontinalis*) was significantly reduced when the fish were exposed to DBP concentrations of 300 µg/L, but not at 90 µg/L. Also McKim reported that 20 µg/L of DBP impairs the reproduction of *Daphnia magna*. The same investigator found that the emergence of adult midges (*Chironomus tentans*) was reduced significantly at a DEHP concentration of 14 µg/L (Johnson *et al.* 1977). However, Streufert *et al.* (1980) exposed a different species of midge, *Chironomus plumosus*, to DEHP through a complete life cycle. Concentrations as great as 360 µg/L did not significantly reduce adult emergence or egg hatchability.

Mayer *et al.* (1977) have shown that adult brook trout and fry of rainbow trout and fathead minnow are unaffected by low concentrations of DEHP even after 150 days of exposure. However, some biological changes in the matrix of the vertebrae were observed. At DEHP concentrations as low as 3.7 µg/L, the collagen content was reduced by 15% in the brook trout.

Effects on Aquatic Plants

Table 14 summarizes 96-h EC_{50} concentrations (concentration at which an effect is noted in 50% of the test subjects) for the three freshwater algae for which data are available. Clearly algae are the most sensitive to BBP, except *Microcystis aeruginosa* which appears to be resistant to this ester. The low EC_{50} value of 110 µg/L obtained with BBP for *Selenastrum capricornutum* is lower than any LC_{50} level found for fish and invertebrate species in Table 13. However, it is still higher than the chronic levels reported for aquatic organisms in the previous section.

The lowest EC_{50} values for DEP and DMP were 85 600 and 39 800 µg/L, respectively. For all three esters tested by the USEPA (1978), similar EC_{50} values with *Selenastrum capricornutum* were found using cell counts and chlorophyll *a* measurements.

Phthalates in Wildlife

There is very little information on any aspect of the presence of phthalates in wildlife. For mammals it is probable that data on laboratory animals, as discussed elsewhere, would be pertinent. There is some information on birds. Peakall (1974) reported that 10 mg/kg of DBP in the diet of ring doves caused a decrease in eggshell thickness and a decrease in shell permeability to water. No effect was produced by DEHP. Belisle *et al.* (1975) fed mallard ducks a diet containing 10 mg/kg DBP or DEHP. No accumulation was found during the five-month study. No residues of DBP could be detected at all, whereas sporadically low levels, about 0.15 mg/kg of DEHP, were detected in the lung and breast. O'Shea and Stafford (1980) fed starlings a diet containing DHP or DEHP at concentrations of 25 and 250 mg/kg for 30 days. They were not

Table 14. PAE Effects on Freshwater Algae

Species	Chemical	Effect	96-h EC_{50} (µg/L)	Source
Selenastrum capricornutum	BBP	Chlorophyll *a*	110	USEPA 1978
S. capricornutum	BBP	Cell number	130	USEPA 1978
S. capricornutum	BBP	Cell number	400	Gledhill *et al.* 1980
S. capricornutum	DEP	Chlorophyll *a*	90 300	USEPA 1978
S. capricornutum	DEP	Cell number	85 600	USEPA 1978
S. capricornutum	DMP	Chlorophyll *a*	42 700	USEPA 1978
S. capricornutum	DMP	Cell number	39 800	USEPA 1978
Microcystis aeruginosa	BBP	Cell number	1 000 000	Gledhill *et al.* 1980
Navicula peliculosa	BBP	Cell number	600	Gledhill *et al.* 1980

Source: USEPA 1980

able to detect any accumulation; both groups of treated animals, however, showed an increased body lipid content. The DEHP-treated group demonstrated a significantly higher body weight gain.

Recommended Limit for Aquatic Organisms and Wildlife

The limited available acute and chronic data for aquatic organisms show the toxicities of PAEs to be species specific and to vary with the ester tested. Acute and chronic levels are as low as 730 and 3 µg/L, respectively, and may be even lower for species more sensitive than those tested. In comparison with the other esters tested, DEHP demonstrates the lowest chronic effects concentration. *Daphnia magna* is the most sensitive freshwater species tested to date.

Employing the chronic toxicity data for daphnids and a safety factor of 0.2, the International Joint Commission (Upper Lakes Reference Group 1977) established recommended maximum levels of 4 µg/L for DBP and 0.6 µg/L for DEHP. It was further recommended that until data became available for other esters, the permissible concentration of the other esters should be restricted to the recommended quantification limit of 0.2 µg/L. The Ontario Ministry of the Environment (1978) has also introduced these limits into its provincial water quality objectives.

In view of the evaluation of the toxicity data presented in this section and the absence of contradictory toxicity levels in the recent literature, the limits outlined above are judged acceptable.

Hence, the concentrations of 4.0 µg/L for DBP, 0.6 µg/L for DEHP and 0.2 µg/L for the other PAEs are proposed as objectives for the protection of aquatic life forms. These recommended objectives will be subject to further review as more information on their toxicity becomes available.

There is insufficient information on which to base an objective for the protection of wildlife.

Water Supply for Livestock

Phthalates in the Animal Body

Most of the research has focussed on laboratory animals and is discussed in the section on drinking water. For domestic mammals the only additional information is that DOP has been found in the pineal glands of cows at concentrations of 700 mg/kg (Taborsky 1967 cited in Leah 1977). As well, DEHP has been reported as being present in the heart of cows (Nazir *et al.* 1971 cited in Peakall 1975).

Some recent data are available for domestic fowl. Jacobs and Ringer (1976) fed female chickens a diet containing 1000, 3000 and 5000 mg/kg of DEHP for four weeks. No effects on body weight, egg production or fertility were noted but food consumption and egg hatchability decreased. Wood and Bitman (1980) fed female chickens a diet containing 1% (10 g/kg) of DEHP for three weeks. Egg production decreased about 20% but no differences in egg weight or other characteristics were noted. In the adults, food intake decreased, plasma lipid and cholesterol levels were lowered about 20%, liver weight and liver lipid and cholesterol content all increased, and the fat content of the pectoralis major muscle decreased. Ishida *et al.* (1981) found traces of up to 0.15 mg/kg of DBP and 0.05 to 0.4 mg/kg of DEHP in the whites, but not in the yolk or shell, of eggs obtained from retail stores. In separate tests, they found DEHP contamination throughout eggs produced by dosed hens.

Based on scanty information it would appear that the types and magnitudes of effects, as well as the metabolic handling of phthalates by livestock, are similar to those found in laboratory animals. However, the information is insufficient to permit the proposal of a recommended level.

Recommended Limit for Livestock

There is insufficient information available to permit the recommendation of a concentration for the protection of livestock.

Water Supply for Irrigation

Phthalates in Terrestrial Plants and Soils

A variety of phthalates have been found in plants and plant products, often in the order of 1 mg/kg, giving rise to speculation that they may be of natural occurrence. However, it is generally unclear whether the phthalates were of natural origin, were accumulated by the living plant from environmental exposure, were introduced in product processing or handling, or were introduced during sampling and analysis. It is evident that further research is required in this area (Peakall 1975; Leah 1977).

Little is known about the toxicity of phthalates to terrestrial plants. At a rate of 1.5 $\mu g/m^2$, DBP in solvent applied to leaves produced chlorosis (yellowing from inhibition of chlorophyll formation) in white mustard (*Sinapis alba*) and milfoil (*Achillea millefolium*). Pure DBP also produced this effect. Nipplewort (*Lapsana communis*) did not exhibit this symptom when treated in a similar manner. The D*i*BP and DEHP did not produce an effect on any of these three plants (Løkke and Bro-Rasmussen 1981).

Phthalates have been found in soil (Whitby and Schnitzer 1978) but data on soil levels are almost non-existent. Persistance in soil, degradation pathways and products, and effects on soil microbiota are all virtually unknown. With reference to the latter points and on the basis of very limited testing, it appears that certain fungi and bacteria do not utilize phthalates as a carbon source. Biosynthesis of phthalates by certain microorganisms has not been unequivocally established (Leah 1977).

Given the nature of their chemical characteristics, it is expected that phthalates would be found associated with the humic component (humic and fulvic acids) in the soil (Whitby and Schnitzer 1978). Adsorption to humic substances increases solubilization of phthalates in water and the complex is an important carrier of phthalates in soil (Khan 1980).

Although there is little evidence at present, it is likely that the two to three orders of magnitude accumulation from water to the bottom sediments in the aquatic ecosystem may occur in a similar manner in terrestrial soil due to binding to humic substances. Until further study, the possibility and nature of soil and subsequent plant accumulation and estimates of any possible consequences would be purely speculative.

Recommended Limit for Irrigation Water

It is not possible to suggest a limit due to insufficient information.

Recreation and Aesthetics

There is no recommended limit.

Industrial Water Supplies

There is no recommended limit.

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APPENDIX

ABBREVIATIONS AND NOMENCLATURE FOR SOME PHTHALIC ACID ESTERS

DMP	dimethylphthalate
DEP	diethylphthalate
D*n*PP	di-*n*-propylphthalate
D*i*PP	di-*iso*-propylphthalate
D*n*BP (DBP)	di-*n*-butylphthalate
D*i*BP	di-*iso*-butylphthalate
DAMP	diamylphthalate
D*n*HP	di-*n*-hexylphthalate
DCHP	dicyclohexylphthalate
D*n*HP	di-*n*-heptylphthalate
D*n*HiP	di-*n*-heptylisophthalate
D*n*HtP	di-*n*-heptylterephthalate
D*n*OP	di-*n*-octylphthalate
D*i*OP	di-*iso*-octylphthalate
DEHP (DOP)	di-(2-ethylhexyl)phthalate[dioctyl phthalate]
D*n*NP	di-*n*-nonylphthalate
D*n*DP	di-*n*-decylphthalate
BBP	butylbenzylphthalate
*n*B*n*DP	*n*-butyl-*n*-decylphthalate
BDP	benzyldecylphthalate
DH*n*NDP	diheptyl-*n*-nonylundecylphthalate

Source: Pierce *et al*. 1980